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AN ESTIMATE OF ADULT MORTALITY IN THE UNITED STATES FROM PASSIVE SMOKING: A RESPONSE TO CRITICISM

Dear Editor:

Lee (1990), Katzenstein (1990), and Holcomb (1990) have commented negatively on my paper (Wells 1988a) in *Environment International*, in which it was suggested that the U.S. death toll from passive smoking may be 46 000 per year. Space does not allow me to deal with all of the points raised, but the more important ones are covered below.

Lee, as tobacco consultants usually do, attacks the underlying studies that I used, particularly the heart studies. I cannot speak for these authors. Dr. Hirayama has written a reply of his own. Dr. Sandler (private communication) has told me that they (Helsing et al. 1988) did look at family size and found no effect. The Johns Hopkins School of Public Health (Helsing et al. 1988) and the University of California, San Diego (Garland et al. 1985) are respected schools of epidemiology, whose researchers presumably know how to adjust for confounding variables. They attempted, within the limits of the data available, to account for known heart risk factors as noted in my paper. What is striking about the heart data in my Table 4 (Wells 1988a) is the consistency of the various results. It is interesting that Lee et al. (1986) made no attempt to adjust for any of the known heart risk factors except age.

Publication bias in smoking studies is an issue often raised by tobacco industry consultants, but so far no one has found a live passive smoking case that is negative. I have dealt with that issue vis-à-vis passive smoking and male lung cancer in my comment (Wells 1988b) on Vandenbroucke (1988). There, it was pointed out that the only available unpublished data were on the high side of the most probable relative risk, not low or negative. In that letter, I asked investigators to send me any data on passive smoking that had not been published or that they had not been able to get published. So far I have received none. As Lee says, the possibility of a large, unpublished data set that found nothing cannot be excluded; it is just extremely unlikely.

For cancers other than lung that are passive smoking related, all except nasal sinus cancer and lung cancer are non-contact sites, as is heart disease. For these sites to be activated, the disease-producing entities must, in most cases, be metabolized and then circulate in the blood and lymphatic systems. Earlier work (Eatough et al. 1986) has shown that 90% of the nicotine in environmental tobacco smoke (ETS) is in the vapor phase. Now Pritchard et al. (1988) have shown that 70% of the tar in ETS is also in the vapor phase. The nicotine and the tar in direct smoking is in the particulate phase. It is true, as Lee says, that smokers are also passive smokers, but for the non-contact sites there is growing evidence that smokers have a higher risk if they are exposed to ETS other than their own than if they are not so exposed. For example, Palmer et al. (1988) found a relative risk for heart disease of 1.34 for spouse exposure of light smoking women and 1.32 for heavy smoking women, and Sandler et al. (1985) found overall cancer risks increasing from unity to 2.4 as active smokers were exposed to an increasing number of household members who smoked. This means that smokers may also be at considerable risk from passive smoking of their own smoke. In other words, for the non-contact sites, the vapor phase tar and nicotine may be the primary culprits, with the particulate phase having less effect. The particulate phase, at least most of it, is relatively quickly cleared. It probably contributes heavily to the contact sites (central lung, mouth, esophagus, and stomach) but then may be eliminated in the feces. All this means that Lee's model for passive smoking, which is based on direct smoking and particulate phase deposition and retention, is likely to predict relative risks for passive smoking that are far too low for the non-contact sites and probably for peripheral lung cancer as well.

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There are other factors that make the prediction of passive smoking health effects by rationing down from the particulate dose of direct smoking chancy. One factor is the possible protective effects in direct smoking. Smoking is known to depress estrogen levels which can protect against breast cancer. Such a protective effect is unlikely from passive smoking. Remmer (1987) postulates that direct smoking activates protective enzymes. Lassila et al. (1988), in their interesting work with monozygotic twins, have shown that direct smoking results in higher levels of prostacyclin, a reactive vasodilator, which, they note, could compensate for the vasoconstrictive effects of cigarette smoking. The dose from passive smoking is probably too low to promote this protective effect. Sinzinger et al. (1982), later confirmed by Burghuber et al. (1986) and Davis et al. (1989), found that platelet sensitivity, a known risk factor for heart attacks, is depressed about 30% in passive smokers, almost to the level found in active smokers. There is no way that the relative retained particulate dose could account for this phenomenon.

Direct smoking and passive smoking are both complex phenomena, with both disease promoting and disease protective components that differ between direct and passive smoking, and where the balance between them differs among individuals. Lee denigrates my suggestion that individual susceptibility could explain, in part, the higher than expected adverse health effects of passive smoking. The science of identifying highly susceptible people is progressing. See for example the work that Caparosa et al. (1989) are doing at the National Cancer Institute on "fast metabolizers" of potential carcinogenic materials. Jones (1986) has shown a substantial difference in sensitivity of different individuals to nicotine and its effect on pulse rate. Khoury et al. (1989) have developed equations for estimating the proportion of persons who are susceptible to a risk factor. They estimate that 13% of smokers are susceptible to lung cancer, whereas only 0.9% of smokers are susceptible to esophageal cancer. My calculations, using their formulae, indicate that only about 0.4% of nonsmokers are susceptible to death by lung cancer from passive smoking.

Lee says that I am "content to include all epidemiological studies" in my meta-analyses, regardless of quality. Actually, I discarded four lung cancer studies because they did not meet stated criteria. The admission criteria are admittedly broad because I did not wish to be accused of biased selection. Originally, I had intended to use only statistically significant data, but the meta-analysis technique allowed the inclusion of smaller studies when properly weighted. A certain amount of scatter is to be expected and is observed in the relative risks from these smaller, low power studies.

Lee (1990) argues that the association between lung cancer and exposure to ETS arises predominately because of bias caused by misclassifying smokers as nonsmokers. In his analysis he seems to have gone out of his way to stretch the data to fit his hypothesis. For example, he states that current typical regular smokers are misclassified to the extent of about 4%. In his workup (Lee 1986, 1987), he has confused smokers who say they are current non-users of tobacco with smokers who say they are never smokers. Yet the epidemiology of passive smoking deals almost exclusively with people who say that they are never smokers. Lee also averages male and female data in order to get higher misclassification factors. Normally in misclassification calculations, one uses sensitivity, which is defined as stated positives divided by stated positives plus false negatives, or in other words, the percent correctly classified as exposed, or in this case, the percent of ever smokers that are correctly classified as ever smokers. By basing his calculations on the number misclassified relative to never smokers instead of relative to ever smokers as he should have, he claims to be able to average male misclassifieds (who are mostly exsmokers) as 18% of self-reported never smokers with female misclassifieds as 6% of never smokers to yield a 10% misclassification factor. The misclassified males as 18% of never smokers are equivalent to only 6% of ever smokers (18% × 25/75) which is essentially the same as the female result (6% × 50/50). Of course the safe thing to do when estimating the bias in female passive smoking relative risks is to use only female data. In a paper in preparation for which I am a co-author, we found, when averaging data from five cotinine studies, including Lee's, that only 1% of female ever smokers said they were never smokers when they were actually current regular smokers, not 4% as Lee contends. Lee uses 10 as the observed relative risk for the regular current smokers that are misclassified as never smokers. The proper procedure is to use smoker relative risks that are consistent with the time frame and locale of the epidemiological studies for which a bias calculation is being made. Fortunately many of the passive smoking epidemiological studies on lung cancer have concurrent estimates of the relative risk of current or ever smokers, and values for the other studies can be estimated from available data. In fact, many of these values are shown on page 72 of Lee's book (1988). A

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log weighted average of the current smoker relative risk for the studies shown in Table 1 of my paper (Wells 1988a) is 4.56 (it was assumed that current smoker relative risk was 30% higher than ever smoker relative risk if those were the only data available) which is less than half the value of 10 used by Lee. Lee's book (1988) has whole sections devoted to misclassification factors for people who say they are recent exsmokers. These data appear to be introduced simply to confuse the reader since they have no bearing on passive smoking epidemiology which deals essentially entirely in self-reported never smokers. If proper factors are used for the extent of smoker misclassification and smoker relative risk, the bias that one calculates agrees with the values previously estimated by Wells (1986a, 1988a) and Wald et al. (1986), and not with those of Lee.

Lee suggests that my estimate of passive smoking deaths may be high. My heart relative risk of 1.23 is supported by two new studies and an update on a third. Palmer et al. (1988) report a female heart relative risk for passive smoking of 1.2, and Humble et al. (1990) report 1.6. Hole et al. (1989), in an update on the study of Gillis et al. (1984), report a female heart relative risk of 2.1 for low exposure passive smokers and 4.1 for high exposure. Sandler et al. (1989) found no increase in risk for total cancer in women, but Miller (1989) in his new study found that non-smoking, non-employed wives of nonsmokers accounted for only 3% of cancer deaths but a much higher percentage of total deaths. These two new results will offset each other. Sandler et al. (1989) also show a statistically significant female all cause relative risk of 1.15 for passive smoking, essentially identical to the 1.165 value I had derived in Appendix B (Wells 1988a) from earlier data. This tends to validate my estimate of 34 000 female all cause deaths from passive smoking. Sandler et al. (1989) also report a statistically significant all cause relative risk for men of 1.17 (the first such data available) that would result in 29 000 deaths per year for a total for both sexes of 63 000, higher than, but not too far distant from the 46 000 deaths that I estimated from the three-disease approach.

In our Western, non-traditional societies, it is very difficult to carry out these low-risk epidemiological studies because of the difficulty of finding a truly nonexposed reference category. Cummings et al. (1990) point out that 91% of the nonsmokers they interviewed had measurable cotinine in their urine while only 76% reported they had been exposed to tobacco smoke in the previous four days. Eighty-four percent of those not living with a smoker had measurable cotinine. If these people are getting nicotine, known to be in the vapor phase of ETS, they must also be getting tar, now known also to be in the ETS vapor phase (Pritchard 1988). Miller (1989) has probably done the best job of ferreting out a nonexposed reference group with the result that he is finding very high relative risks for total cancer from passive smoking.

As Goldstein (1986) has said, "Chemicals shown to be carcinogenic are considered by regulators as 'guilty until proven innocent' of having no threshold. This conservative approach essentially puts the burden on the producer or user of providing the scientific evidence justifying a threshold in regulating a carcinogen." The purpose of my paper was to provide regulators with an estimate of the most probable death toll from passive smoking given the existing epidemiological evidence, and also data from which to calculate an upper bound estimate, as they usually wish to do. Nothing in Lee's comments, with his botched bias analysis and his flimsy dose model, does anything to "justify a threshold" for this known human carcinogen.

Katzenstein (1990) also appears to be very selective in the data that he reports in Table 1 of his letter and he does not appear to have done his homework in finding all the reports on passive smoking and lung cancer that have issued since the 1986 reports of the National Academy of Sciences (NRC 1986) and the Surgeon General (USSG 1986). Commenting first on the reports that he lists, Chan and Fung (1982) is simply a restatement of the more detailed data in Chan et al. (1979). I had rejected Chan et al. (1979) and Dalager et al. (1986) for reasons stated in my paper. Dalager's crude relative risk of 1.00 that Katzenstein reports is for both sexes. The only female all exposure relative risk in that paper is 1.96 for spouse exposure, not statistically significant. However among older women, 63 plus years of age, with high intensity exposure, the odds ratio was 5.14 with 95% confidence limits of 1.4 to 18.95. A dose response trend was also observed. Kabat et al. (1984) found a statistically significant odds ratio of 3.3 for male exposure at work and also found a statistically significant Mantel test for linear trend in the frequency of exposure (four levels) for males ($p < 0.005$). Garfinkel et al. (1985) had a statistically significant odds ratio of 2.0 at the highest exposure. The results that Katzenstein quotes from Gao et al. (1987) are for never smoking women who ever lived with a smoker. For spouse exposure they report a rising relative risk from 1.0 for less than twenty years exposure to a statistically significant 1.7 for forty

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plus years exposure. Shimizu et al. (1988), besides reporting the 1.1 nonsignificant risk for nonsmoking wives exposed to a husband's smoke also report a 4.0 significant risk for exposure to a mother's smoking and 3.2 for exposure to the husband's father's smoking. The latter is not unusual since wives in Japan, after they leave their mother's home, often live with the husband's family and the husband's father is often retired. Wu et al. (1985), Brownson et al. (1987), Humble et al. (1987), and Lam et al. (1987) were covered in my paper (Wells 1988a). The male relative risk in Humble et al. (private communication) is a statistically significant 4.2. New reports that Katzenstein evidently is not aware of are (1) the Hong Kong thesis of W. K. Lam (1985) with 60 female cases and a statistically significant relative risk of 2.01 and a risk for peripheral tumors of 2.64 ($p < 0.05$); (2) Geng et al. (1988) with 54 cases and a statistically significant odds ratio of 2.16 for all levels of exposure, and 2.76 with 95% confidence limits of 1.85 to 4.10 for exposure to 20 plus cigarettes per day. They also report a relative risk from ETS for smoking wives of 1.88; (3) Inoue and Hirayama (1988) with 22 cases report a nonsignificant odds ratio of 2.25 for all exposure levels, but for exposure to 20 plus cigarettes a day the odds ratio is a statistically significant 3.35 (they also report a statistically significant positive trend); (4) Svensson (1988), in a thesis from Sweden, with 34 female nonsmoking lung cancer cases, found a relative risk of 1.2 for exposure at home or at work and a relative risk of 2.1 for exposure at home and at work. He also found a relative risk of 1.4 for exposure as a child or as an adult and 1.9 for exposure both as a child and as an adult. None of Svensson's relative risks is statistically significant; and (5) Varela (1987) also in a thesis, this one from Yale University, reports on 218 female cases and 221 male cases which included both never smokers and long-term exsmokers. He found no increase in risk for spouse exposure or workplace exposure but found a statistically significant relative risk of 1.87 multiple exposures at home.

Katzenstein's attack on the underlying studies is a typical tobacco industry approach. As we know, all epidemiological studies are flawed to one extent or another. However the National Academy and the Surgeon General, looking at the totality of the studies then available, concluded that passive smoking can cause lung cancer, and inclusion of the studies new since 1986 would not change that conclusion.

Katzenstein is wrong when he says that the heart studies failed to consider cardiovascular risk factors. Garland et al. (1985) and Helsing et al. (1988) adjusted for several of them. The Svendsen study (1987) considered ten of the most frequently studied heart risk factors, comparing 286 nonsmoking men married to smokers and 959 married to nonsmokers. The differences were small, and adjusting for them did not decrease the observed risk. Katzenstein quotes an American Cancer Society 1988 release saying that currently available evidence is not sufficient to conclude that passive or involuntary smoking causes lung cancer in nonsmokers. He must have found this in the rare book store since neither the Delaware office nor the national office of the American Cancer Society could find this reference. On the contrary the ACS "Cancer Facts and Figures for 1989" states that involuntary smoking increased the risk of lung cancer, and their "The Smoke Around You" pamphlet issued in 1987 quotes the 35% increase in lung cancer risk for passive smoking that is found in the National Academy report (NRC 1986).

In Katzenstein's "final comment" where he quotes the NAS and USSG reports on passive smoking and heart disease, he fails to note that the best heart evidence is in papers issued since those reports came out. It is interesting that the newest reports (Palmer 1989; Hole 1989; Humble 1990) all support a positive relative risk.

Holcomb (1990) states that I had encouraged the view that the results in Wells (1988a) were new. Actually that paper has a long history. The original version was presented at a seminar at the Harvard School of Public Health in December, 1984. An update was presented to the National Research Council in January, 1986. The version Holcomb refers to was presented at the June, 1986, meeting of the Air Pollution Control Association, and in September, 1986, before the Natural Resources, Agriculture Research and Environment Subcommittee of the Committee on Science and Technology of the U.S. House of Representatives. It is published in the proceedings of those meetings (Wells 1986b, 1987). After extensive revision, a shortened version was presented at the 6th World Conference on Smoking and Health in Tokyo in November of 1987. A summary is published in the proceedings of that meeting (Wells 1988c). The first draft of the current version (Wells 1988a) contained a summary of this history, but the editors of *Environment International* decided that since none of the earlier versions had been adequately peer reviewed, reference to them could be omitted. It should be noted that the current paper profited by the many comments received over the years from many experts in the field who either commented gratuitously or whose advice was solicited. James Repace was sur-

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prised in 1986 at the large number of heart deaths and is probably still surprised, as are many others, but that is the way the numbers come out.

Holcomb states that I did not address the issue of causation. Perhaps this should have been done more explicitly in the paper. It was pointed out on the first page of the paper that the Surgeon General's report (USSG 1986) and the National Academy report (NRC 1986) both stated that passive smoking can cause lung cancer. I thought that was adequate coverage for that issue. (Incidentally Holcomb states that "the Surgeon General's report was alone in concluding that ETS causes lung cancer in nonsmokers," but on page 10 of the National Academy report it is stated, "Considering the evidence as a whole, exposure to ETS increases the incidence of lung cancer in nonsmokers.") Then I went on to show that the heart data, including the new data, had most of the same characteristics as the lung cancer data in terms of number of cases, statistical significance, dose response, and biological plausibility. Hence one could infer causation.

Holcomb references a paper by Koo et al. (1988) that allegedly shows that nonsmoking women married to ever smokers had a less healthy life style than nonsmoking women married to nonsmokers. Careful analysis of their voluminous data indicates eight life style factors where the test p and the p for trend were both reasonably small. Five indicated a healthier life style for the women married to the never smokers and three for those married to the smokers. About all this paper shows is that nonsmoking women in Hong Kong who lived in rural areas are more likely to be married to nonsmokers and to have a more rural life style. Humble et al. (1990), in their soon-to-be-published paper on passive smoking among never smoking women in Georgia, found that higher social status white women had a higher relative risk of heart disease from ETS than lower social status white women, quite the reverse from what Koo et al. concluded. Humble et al. also adjusted for age, diastolic blood pressure, total serum cholesterol, and body mass. The tobacco people have used misclassification as their principal smoke screen to discredit lung cancer risk from passive smoking. They know that misclassification can't possibly explain the heart effects of passive smoking so they have embraced "life style". This also is proving to be ephemeral.

Holcomb complains that I included unpublished studies in the analysis, but Katzenstein complains that publication bias is likely to omit pertinent data. I chose to include all the data I knew about, favorable or unfavorable. Omission of the unpublished studies would not change the conclusions.

Holcomb states that I based my exposure estimates on data published by Freidman et al. (1983). Actually, the exposure of never smokers living with ever smokers was obtained from the exposure of controls reported in the various U.S. passive smoking studies. This represents the major factor in female exposure. Only the exposure of nonsmokers living with nonsmokers was estimated using Friedman et al.

There is no question that my conclusions on heart disease and cancers other than lung go further than the cautious statements in the Surgeon General and National Academy of Science reports. So far, however, the new data support my position. Whether causation has been "proved" or not, public health officials need to know the mortality stakes involved. They can then make their own judgments as to the likelihood of causality.

Holcomb has not read the paper of Repace and Lowrey (1985) carefully. Their estimate of 4665 lung cancer deaths from passive smoking is based on a comparison of lung cancer mortality rates of Seventh Day Adventists who never smoked with those of non-Seventh Day Adventists who never smoked, not on exposure estimates as Holcomb claims. My estimates in no way rely on the exposure estimates of Repace and Lowrey. There are some nine studies in the literature that estimate lung cancer deaths from passive smoking. Except for Arundel et al. (1987) estimate, they range from 600 to 5600. The Arundel estimate is based on extrapolation from smokers to nonsmokers of retained particulate dose, an idea discredited earlier in this letter.

As Katzenstein says, death from passive smoking is a serious issue, serious to the health of the tobacco industry, and serious to the public health. We can expect vigorous (but misleading) attacks from the tobacco side, as these three letters show, but it is still best to lean toward safety when the health of the public is at stake.

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